Immediate Concerns

Effective CPR can be performed by following a few basic rules:

- Immediately assess the environment for danger and move the patient if necessary. Never assume that an environment is safe.
- Minimize the time from cardiac arrest recognition to start- ing effective CPR. For every minute without CPR during witnessed VF CA, survival decreased by 7% to 10% in con- trast to a decrease in survival by 3% to 4% per minute when bystander CPR preceded attempted defibrillation (6).
- Defibrillate immediately if a defibrillator is rapidly available (<3–5 minutes) in patients with VF. This should be the pri- mary treatment focus within the first few minutes of SCA due to ventricular fibrillation. For each minute delay in de- fibrillation, chances of eventual hospital discharge decreased by 8% to 10% (7). Note that if the time from arrest to emer- gency medical service (EMS) arrival and initiation of CPR is more than 3 minutes, provision of 2 minutes of CPR be- fore attempted defibrillation was associated with improved outcome (8,9).
- “Push hard and fast” during chest compressions and min- imize the duration of interruptions to reassert the patient’s rhythm. It is recommended to interrupt chest compressions only briefly and about every 2 minutes to assess the rhythm and switch rescuer if feasible.
- While CPR is in progress, attempt to identify the cause of arrest. Although the steps of CPR are uniform, other resus- citation interventions may be indicated based on the cause of CA. If the patient fails to respond to standard CPR inter- ventions, think about delayed recognition and recall the H’s and T’s (Table 46.1) to help identify a potentially correctable cause for ongoing cardiac arrest.
- Good teamwork increases the effectiveness of resuscitation when more than one rescuer is available.
- Attention to postresuscitation care is an important element of neurologic outcome. Focus on restoring and supporting adequate cardiac output and tissue perfusion, monitor and maintain normal blood glucose concentrations, treat the un- derlying cause of the arrest, maintain normothermia, and consider therapeutic hypothermia to maximize survival and cerebral recovery.
- If the patient fails to respond to effective CPR, appropriate judgment is needed in determining when to stop resuscitative efforts. Guidelines for CPR and advanced adult and pediatric advanced life support are published every 5 years by the American Heart Association (AHA) (10). These guidelines are based on an exten- sive evidence-based review by national and international ex- perts leading to the publication of a consensus on science and treatment recommendations prepared by the International Li- aison Committee on Resuscitation (ILCOR) (11,12). ILCOR is a collaborative organization of American, European, Aus- tralian, Canadian, South African, and Latin American resusci- tation councils with the goal of identifying the best resuscita- tion evidence (13).

Table 46.1

<table>
<thead>
<tr>
<th>POTENTIAL CORRECTABLE PROBLEMS DURING CARDIAC ARREST: “6 HS AND 5 TS”</th>
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<tr>
<td>Hypovolemia</td>
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<td>Hydrogen ion (acidosis)</td>
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<td>Hypo/hyperkalemia</td>
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<td>Tension pneumothorax</td>
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<td>Thrombosis, coronary or pulmonary</td>
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<td>Trauma</td>
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Outcome of Cardiopulmonary Resuscitation

Measuring Outcome after Cardiac Arrest

Retrospective and prospective observational outcome reports have been published about the success rates of both in-hospital and out-of-hospital cardiac arrest, but most of these are single-center reports with relatively few subjects. More recently, cardiac arrest events and outcomes have been systematically monitored in large multicenter settings. To facilitate data comparison among communities, international guidelines were pub- lished for data collection using standard definitions of events and outcome for both adults and children (the Utstein Guideline- lines) (14–16). In 2000, the AHA established a National Reg- istry of Cardiopulmonary Resuscitation (NRCPR) to system- atically collect data on the epidemiology, processes of care, and outcome of in-hospital CPR (4,17). This comprehensive database has, at the time of this writing, an excess of 100,000 cardiac arrest events and provides important insights into the mechanism, logistics, and outcomes of cardiac arrest and helps evaluate the impact of interventions such as a rapid response team. The standard consensus terms used to describe outcome of CA are as follows (13):

1. Successful defibrillation: Termination of fibrillation to an organized electrical rhythm (including PEA for at least 5 seconds after shock delivery).
2. Return of spontaneous circulation (ROSC): Restoration of a spontaneous perfusing heart rhythm in the absence of ex- ternal chest compressions for approximately >30 seconds. This term is not applicable when extracorporeal support is used, such as extracorporeal membrane oxygenation or a biventricular assist device.
3. Survived event:
   a. Out-of-hospital setting: Sustained ROSC with spontane- ous circulation until admission and transfer of care to medical staff at the receiving hospital.
   b. In-hospital setting: Sustained ROSC for >20 minutes (or support of circulation if extracorporeal circulatory sup- port is applied).
4. End of event: A resuscitation event is deemed to have ended when either death is declared or spontaneous circulation is restored and sustained for 20 minutes or longer. If extracorporeal life support is being provided, the end of event is 20 minutes after the establishment of extracorporeal circulation.

5. Survival to hospital discharge: Survival to hospital discharge is the point at which the patient is discharged from the hospital acute care unit regardless of neurologic status, outcome, or destination.

6. Neurologic outcome at discharge: Assessment of neurologic recovery is measured at discharge in terms of recovery of consciousness and achievement of precardiac arrest level of functioning.

Out-of-hospital Cardiac Arrest

Despite improvement in the scientific basis for resuscitation practices and extensive efforts at CPR training of lay and professional rescuers over the years, the outcome of most adult victims of out-of-hospital cardiac arrest remains poor, with a median reported survival to hospital discharge of 6.4% (7,18). A recent study suggested that adults who had a cardiac arrest in a public location (i.e., witnessed) were more likely to arrive to the hospital alive (39% vs. 31%, p = 0.049) and were more likely to have a good neurologic outcome after 6 months (35% vs. 23%, p = 0.023) compared with patients who had a cardiac arrest in a nonpublic location (19).

In children, the epidemiology and physiology of OOH cardiac arrest are different. In this group of patients, a recent systematic review of 41 studies of out-of-hospital cardiac arrest, including trauma, revealed an ROSC of 30%, with survival to admission of 24% but survival to discharge of 12% and neuroligically intact survival of only 4% (20). The initial cardiac rhythms observed in these children were asystole, 78%; PEA, 12.8%; VF/pulseless VT, 8.1%; and bradycardia with a pulse, 1%.

In-hospital Cardiac Arrest

Despite efforts to train professional in-hospital rescuers to deliver effective CPR and use various devices and therapeutic drugs during resuscitation, the objective survival rates over the years have hardly changed (21,22). Currently, in-hospital cardiac arrest in adults has an overall survival of about 18% (17,23–25). Analysis of data from the NRCPR found that the prevalence of VF or pulseless VT as the first documented pulseless rhythm during in-hospital cardiac arrest was only 21% in adults and 14% in children (4). The prevalence of asystole as the initial rhythm was 35% in adults and 40% in children, whereas the prevalence of PEA was 32% versus 24% in adults and children, respectively. The rate of survival to hospital discharge following pulseless cardiac arrest was higher in children than adults, 27% versus 18%, respectively. Of these survivors, 65% of children and 73% of adults had good neurologic outcome. After adjusting for known predictors, such as arrest location and monitoring at time of arrest, outcome was surprisingly worse when the rhythm was VF/VT in children compared with asystole and PEA (4). Further analysis of these data showed that VF/VT occurred during CPR in children more commonly than it occurred as the initial rhythm (26). Survival to discharge is highest (33%) when VF/VT is the initial rhythm compared with much worse survival (11%) if this rhythm develops during resuscitation (26).

Neurologic Outcome

Neurologic outcome is determined by the cause of arrest (e.g., degree of shock or hypoxemia prior to arrest), the duration of no flow, adequacy of flow during CPR, restoration of adequate flow following ROSC, and subsequent injury secondary to postarrest management such as the occurrence of hyperthermia or hypoglycemia. Survivors who ultimately have a good outcome generally awaken within 3 days after CA. Most patients who remain neurologically unresponsive due to anoxic-ischemic encephalopathy for more than 7 days will fail to survive, and those who do survive often have poor neurologic recovery (27–29). Neurocognitive impairment ranges from dependency on others for care to remaining in a minimally conscious or vegetative state. Achieving good functional outcome is the ultimate goal for successful CPR because survival with severe neurologic injury is a terrible cost to patients, their family members and friends, and society at large. The financial implications of caring for these patients with disorders of consciousness are substantial (30).

Unfortunately, most studies reporting outcome data lack these difficult-to-obtain elements or have used crude methods to describe neurologic outcome, such as the composite scores from the Glasgow outcome scale (GOS) (31) and Cerebral Performance Category (CPC) (32,33). One important limitation of these scales is the possibility of wide variation of neurologic function for the same score. In children, other scales such as the Pediatric Cerebral Performance Category (PCPC) and Pediatric Overall Performance Category (POPC) have been used (34–36). Currently, 11% to 48% of CA patients admitted to the hospital will be discharged with good neurologic outcome (21,37,38). Recent data from the NRCPR show that neurologic outcome in discharged adult survivors is generally good with 73% of patients with Cerebral Performance Category 1 (17).

INITIAL CONSIDERATIONS

CPR is primarily based on two principles:

1. Providing artificial ventilation and oxygenation through an unobstructed airway to maintain gas exchange. Since cardiac output is limited, the rescuer needs to avoid ventilation in excess of that required for adequate ventilation/perfusion matching.

2. Delivering chest compressions to maintain threshold blood flow, especially to the heart and brain, while minimizing interruption of compressions.

Basic Life Support

Basic life support (BLS) is the initial phase of CPR recognized as the “ABCs” of CPR: A, airway; B, breathing; C, circulation. Effective BLS can provide almost 30% of normal cardiac output with adequate arterial oxygen content; this is sufficient to protect the brain for minutes until effective defibrillation or
other definitive therapeutic maneuvers are provided (39). With
the 2000 guidelines for CPR, a new evidence-based approach to
ventilation during CPR was introduced that continued with the
2005 edition. New evidence from laboratory and clinical sci-
ence led to a de-emphasis on the role of ventilation following a
dysrhythmic cardiac arrest (arrest primarily resulting from an
acute cardiovascular event, such as ventricular fibrillation or
tachycardia) (40). However, after asphyxial cardiac arrest (car-
diac arrest primarily resulting from respiratory arrest and less
commonly from shock), the traditional emphasis on ensuring
airway patency, effective ventilation, and circulation remains
fundamental to achieving survival and the intact neurologic
outcome of patients. The following section summarizes BLS in
adults; differences in BLS for infants and children compared
with adults are seen in Table 46.2.

Airway

After establishing unresponsiveness and calling for help, open-
ing the airway and maintenance of airway patency are the next
steps of CPR. Provision and maintenance of a patent upper air-
way should be provided with the maneuvers listed below. These
maneuvers are described in detail in basic airway management
chapters.

1. Head tilt/chin lift
2. Jaw-thrust maneuver
3. Triple airway maneuver
4. Mandibular forward displacement

If airway obstruction is felt to be present and foreign body
is suspected, immediate removal should be done using basic
airway management guidelines.

Breathing

Determination of Apnea. There are several recommended
methods of determining the absence of spontaneous breathing
in an unconscious patient when no pulse oximetry is present.
Spontaneous respiration may be difficult to observe unless the
airway is opened (41). Once airway patency is established, the
rescuer should place his or her ear over the victim’s mouth
and nose. The rise and fall of the chest should be noted while
listening for breath sounds and feeling for expired air. The ab-
sence of these signs is indicative of apnea. Frequently, gasping,
grunting, snoring sounds or agonal respiratory efforts may be
observed during sudden cardiac arrest, especially within the
first few minutes of collapse. This is typically not sustained,
and should not be mistaken for adequate spontaneous respi-
ration. In some instances, the victim may exhibit respiratory
efforts but no air exchange is observed. This indicates severe
upper airway obstruction; opening the airway will facilitate the
resumption of air movement. Despite the widespread diffusion
of these recommendations, studies showed that lay rescuers as
well as professional rescuers are unable to accurately determine
adequate breathing in unresponsive patients (42,43). Currently,
it is recommended that if breathing cannot be confirmed within
10 seconds, two rescue breaths should be given.

Initiation of Rescue Breathing. If the patient is not breathing,
rescue breaths are delivered using the maneuver app-
propriate for the situation. Most first-responder personnel
and medical professional staff in hospitals are trained to use adjunct
devices during BLS. When a bag-mask ventilation (BMV) is not
immediately available, mouth-to-mouth rescue breathing can
still be life saving.

Intuitively, the presence of vomitus and fear of infectious
contamination can affect the willingness of rescuers to perform
mouth-to-mouth resuscitation. Lawrence and Sivaneswaran
showed that only 13% of 70 hospital staff members surveyed
would prefer to do mouth-to-mouth ventilation during CPR and
59% would prefer to do mouth-to-mouth ventilation (44). Specialized
breathing masks with a one-way valve can be utilized during
CPR. The valve prevents the exhaled air from entering the res-
cuer’s mouth.

During both one and two-rescuer CPR, two mouth-to-
mouth breaths are given during a pause after every 30th chest
compression. This ratio is the same for adults, children, and
infants when there is only one rescuer. If two or more pro-
fessional rescuers are present, a 15:2 ratio is used in children
(defined as child if there are no signs of puberty such as facial
hair or breast development) and infants.

Once an advance airway (i.e., endotracheal tube, laryngeal
mask airway [LMA], or pharyngotracheal Combitube) is ob-
tained, chest compressions are given continuously at a rate
of 100 compressions per minute without interruption for venti-
lation. A ventilation rate of eight to ten can sufficiently match
the perfusion achieved by chest compression (20%–30% of
normal at best) (45). Rescue breathing when there is a pulse
should not exceed 12 breaths/minute (45).

Manual Resuscitators (Bag-valve Devices). Bag-valve devices
(BVDs) of various designs and sizes are available for adults and
children to deliver manual breaths. A self-inflating, manually
operated bag with a nonbreathing valve is preferred because it
allows ventilation even if there is no connection to an oxygen
source. This device may be used in conjunction with a face
mask, endotracheal tube, or other invasive airway device.

The standard parts of a BVD include (a) a delivery port
with a 15-mm/22-mm adapter coupling that can be connected
to the mask or tracheal tube; (b) a one-way, non-jam valve that
allows a minimum of 15 L/min oxygen flow rate for sponta-
neous and controlled ventilation; and (c) a system for ensuring
delivery of a high oxygen concentration through an auxiliary
oxygen inlet at the back of the bag or by an oxygen reservoir.

The bag holds a volume up to 1,600 mL. Some pediatric re-
suscitator bags are provided with a 25- to 30-cm H2O pop-off
valve to avoid excessive positive airway pressure, thereby redu-
ing the risk of gastric insufflation and hyperinflation of the lungs
and subsequent pulmonary barotrauma, but a pop-off valve is not recommended during CPR in adults since higher
airway pressures may be needed to achieve adequate ventila-
tion. A malfunctioning valve may lead to improper venting and
inadequate ventilation. This can be recognized by the sound of
air escaping through the relief valve while squeezing the bag.
Adjusting or partially occluding a malfunctioning valve may
be life saving.

The operator should be positioned at the top of the victim’s
head. Appropriate face mask size should be selected, the air-
way opened, and a tight seal created covering the mouth and
the nose as described previously. After proper head positioning,
the bag is squeezed to deliver the breath over 1 second. Tidal
volume is sufficient if visible chest rise is present. An orpha-
nygeal airway or nasopharyngeal airway may be used to facil-
itate bag-mask ventilation in case of upper airway obstruction.
Proper bag-mask ventilation requires training, practice, and
familiarity with the equipment.

A single rescuer may have difficulty maintaining a correct
head position and delivering adequate tidal volume at the same
time (46–48). Thus, two rescuers may be more effective where
one rescuer seals the mask to the mouth, performs a jaw thrust,
and maintains head extension while the other rescuer squeezes
the bag with both hands (49). Oxygen supplementation during
bag-mask ventilation delivers 40% to 60% O2 without an oxy-
gen reservoir and over 90% with an oxygen reservoir (50,51).
Complications associated with the use of a bag-mask de-
vice are primarily due to excessive ventilating pressures caus-
ing pneumothorax, pneumomediastinum, and gastric disten-
tion that can lead to decreased total lung compliance, regurgi-
tation, and gastric rupture (52). Unless the rescuer is proficient
in the use of flow-inflating devices (e.g., Mapleson D system,
typically used by anesthesiology personnel during CPR), it is pref-
erable to use a self-inflating handheld resuscitator bag.

Special consideration should be applied for pediatric pa-
ients. In fact, the pediatric airway may be flattened by exces-
sive extension of the cervical spine during the head tilt-chin
lif maneuver. Smaller breaths must be used to avoid abdom-
inal distention, regurgitation, and pulmonary barotrauma. As
with adults, appropriate tidal volume is characterized by visible
chest rise.

Oxygen-powered, Manually Triggered Ventilation Devices.
Oxygen-powered breathing devices may be used when avail-
able during CPR. These simple ventilatory devices require high
oxygen flow rates to overcome air leak (44). Although they may
be able to deliver adequate tidal volumes, high-flow-rate time-
cycled oxygen-powered devices carry a risk of severe gastric
distention when used with a face mask.

Inspiratory Impedance Valve. A small inspiratory impedance
valve device was recently introduced to occlude the airway se-
lективally during the decompression phase of chest compressions
without interfering with exhalation or resuscitation bag venti-
lation. This device, as shown in Figure 46.1, is placed between
the end of an endotracheal tube or face mask and the resus-
citation bag, which then limits air entry into the lungs during
chest recoil or when the patient spontaneously breathes. In the
latter circumstance, the patient needs to overcome the inspiratory threshold pressure for air flow to occur. The resulting modest reduction in intrathoracic pressure enhances venous return to the heart between chest compressions and thus cardiac output. This device has no deleterious effect on ventilation, and can enhance coronary and cerebral blood flow during CPR in animals and humans along with a higher rate of ROSC after defibrillation (53,54). An enhancement of cardiac output during spontaneous ventilation has also been observed in hypovolemic shock states (55). In spite of the favorable hemodynamic effect, the use of this device so far has not demonstrated a definitive long-term neurological improvement after ROSC. At the time of this writing, a large multicenter clinical trial is under way.

Mechanism of Blood Flow during Chest Compression. In the early 19th century, open cardiac massage was used successfully by Kristian et al. in the emergency treatment of cardiac arrest (59). In a short time, this became the method of choice for CPR (60,61), with variable outcomes reported. Open cardiac massage requires a thoracotomy and the heart is squeezed directly by the hands of the rescuer (61,62). The basic idea was to produce an effective perfusion pressure gradient for blood flow from the heart to vital organs. Open cardiac massage has been utilized in a variety of special situations where the access to the chest is immediately feasible (63–66). For example, in the cardiac intensive care unit (ICU), open cardiac compression is used both in adult and pediatric patients in the immediate postoperative setting (67). Outside of the operating room (OR), the
use of open cardiac massage, although it improved coronary perfusion pressure and increased ROSC, was not followed by improved survival (68).

In 1960, Kouwenhoven et al. reported successful resuscitation of dogs and subsequently humans with ventricular fibrillation using the combination of closed chest compression, artificial respiration, and electrical defibrillation (69,70). They hypothesized that the heart was physically squeezed between the sternum and vertebral column, whereby the blood flow generated is similar to the mechanism of spontaneous contraction of the heart; hence, this is called the “cardiac pump” model. This model, however, does not explain several clinical observations in contrast with the cardiac pump theory such as the ineffectiveness of CPR during flail chest, although theoretically it should be easier to compress the heart in this condition, or the effectiveness of closed chest CPR in patients with hyperinflated chest due to severe emphysema.

The cardiac pump model was challenged in the 1970s when Criley et al. reported ROSC with cough during ventricular fibrillation (71,72). In 1980, Rudikoff et al. reported that fluctuations of intrathoracic pressures were primarily responsible for blood flow during CPR (73,74). These findings supported a noncardiac or “thoracic pump” mechanism for blood flow during CPR. This model proposes that increased intrathoracic pressure during chest compression elevates the pressure of blood located in structures within the thorax, creating the gradient for forward blood flow from the intrathoracic to lower pressure extrathoracic arteries. During relaxation, intrathoracic pressure drops, resulting in refilling of the heart with blood. In this model, the heart acts as a passive conduit. Echocardiographic studies to elucidate the pumping mechanism during cardiac arrest have failed to resolve the controversy (73–77). It appears that in adults with thin chest walls, direct cardiac compression does occur, whereas in prolonged resuscitation and in patients with thick chest walls or hyperinflated lungs, the thoracic pump mechanism becomes the predominant flow mechanism. It is also likely that both mechanisms are involved in some cases.

In infants and children, a mechanism of direct cardiac compression is well supported by the observation of CPR performed in young animal models (78). Currently, closed chest compression is the standard method of producing blood flow during CPR for both adult and pediatric victims.

Closed Chest Compression. Closed chest compressions consist of the rhythmic application of pressure over the lower half of the sternum. Even if properly performed, in adults, chest compressions can only produce systolic arterial pressures of 60 to 80 mm Hg; diastolic pressure is usually low and mean arterial pressure in the carotid artery seldom exceeds 40 mm Hg (79). These values are low compared to normal but still can provide critical blood flow to the heart and brain while attempts to restore definitive cardiac activity are implemented. To achieve maximum benefit from chest compressions during CPR, attention should be given to the following components:

1. Positioning the patient. The victim should lie supine on a hard surface. If the patient is on a soft bed, then place a backboard beneath the patient (80). The rescuer should kneel beside the victim’s thorax if the victim is on the floor (81), or adjust the bed to the level of the rescuer’s hip if the victim is on a bed. Other positions like over-the-head CPR and two-person straddled CPR are described, but the efficacy of these techniques has not been established (82).

2. Positioning of rescuer hands. In adults, the rescuer ideally should compress the lower half of the victim’s sternum. Locating proper hand position, however, has been confusing in lay rescuer education, so it is easier to teach that the rescuer should place the heel of the hand on the sternum in the center (middle) of the chest between the nipples and then place the heel of the second hand on top of the first so that the hands are overlapped and parallel (83,84). Compressions are delivered while keeping the arms straight at elbow. This is best achieved by leaning over the victim, which allows the rescuer’s weight to be used in producing effective chest compression, but the rescuer should avoid maintaining compression on the chest during the relaxation phase, since this impairs venous return (85,86).

In children, the rescuer again should ideally compress the lower half of the sternum, avoiding the xiphoid process and the ribs, but the lay rescuer is taught to position the heel of one hand at the midinnipple line. Either one or two hands can be used in children, depending on the strength of the rescuer. In a child, the use of two hands generates higher compression pressures compared with one hand and is less fatiguing in a manikin study (87), but no outcome study has shown superiority of one method over the other.

In infants, two different techniques are used. The two-finger technique and the two-thumb encircling technique. With the two-finger technique, the rescuer compresses the lower half of the sternum just below the intermammary line with the second and third or third and fourth fingers (78,88–90). This is the only hand position to be used by lay rescuers. The two-thumb encircling hands technique is recommended when there are two health care provider rescuers. The infant’s chest is encircled with both hands with the thumbs placed on the lower half of the sternum and the fingers spread around the thorax. Compression is delivered with both thumbs while the lateral chest is squeezed simultaneously with the fingers. The two-thumb encircling technique produces more consistent compression depth and higher coronary perfusion pressures (91–93).

3. Depth of compression. The sternum is depressed approximately 1 1/2 to 2 inches (approximately 4 to 5 cm) in adults. In children and infants, the chest should be compressed to achieve one-half to one-third the depth of the anterior-posterior diameter of the chest. The higher proportional depth of compression is appropriate in infants, whereas one-third the depth of compression is appropriate in children.

Data in manikin models show that rescuers often do not sustain an adequate depth of compression during CPR for more than 2 minutes. In studies of adults undergoing CPR in both the out-of-hospital (94) and in-hospital (95) setting, inadequate compression depth is commonly observed. Since adequate depth of compression is important in achieving optimal forward flow, emphasis should be placed on compressing “hard” as well as fast enough, and the rescuer performing chest compressions should change approximately every 2 minutes during the time when the patient’s rhythm is being reassessed.

4. Compression rate. Recent studies in animals (96) and in humans (97,98) support that a rate of at least 80 per minute should be achieved to provide optimal forward blood flow during CPR, with some experts suggesting higher cardiac
output with compression rates up to 150 per minute. Higher compression rates, however, are more difficult to sustain. It is important to remember that the compression rate refers to the speed of compressions, not the actual number of compressions delivered per minute. The actual number of chest compressions delivered per minute is determined by the rate of chest compressions and the number and duration of interruptions to open the airway, deliver rescue breaths, and time for analysis of cardiac rhythm and delivery of shock. To maximize the beneficial effects of chest compressions producing blood flow, the 2005 guidelines recommend compressions at a rate of approximately 100 per minute with interruptions in chest compressions kept to a minimum.

5. Decompression: After achieving the maximum depth of compression, the chest should be released to recoil on its own. It is crucial to let the chest recoil completely before the next compression is started. Incomplete chest recoil leaves residual pressure (relative to atmospheric intrathoracic pressure that decreases both coronary and cerebral perfusion (86).

6. Duty cycle: Duty cycle refers to the sum of the time interval from the start of compression until maximum compression is achieved (i.e., “compression cycle”) plus the time interval from the maximum compression to complete decompression (i.e., “decompression cycle”) prior to the next compression. Studies in animals and in manikin models showed that coronary and cerebral perfusion directly increased with increasing compression rate up to 130 to 150 per minute using a compression cycle between 20% and 50% of the duty cycle (99,100). Current guidelines recommend a 50% compression cycle (101).

Compression-to-Ventilation Ratio during Basic Life Support

Differing compression-to-ventilation (C/V) cycles were recommended when one or two rescuers are present. In children, additional ratios were used to provide more ventilation. Recent studies show that both lay and health care rescuers provide poor chest compressions (94,95), and there is often a prolonged hands-off period during CPR while breaths are being given or the rhythm is being assessed, resulting in no cardiac output. Both animal data and theoretic models suggest that higher cardiac output will be achieved using a higher ratio of chest compressions and fewer interruptions for ventilation. The latter is acceptable since the overall cardiac output is only 25% to 30% of normal at best, which means that ventilation only needs to be 25% to 30% of normal to achieve adequate matching of ventilation to cardiac output during CPR. Therefore, the new guidelines recommend one universal ratio of 30 compressions to two ventilations in adults, children, and infants with one rescuer. If two health professionals are present, they should use a 15:2 ratio in children and infants. This approach maximizes chest compressions and minimizes time for ventilation, rhythm analysis, and shock, and improves ROSC and outcome (9,102).

In addition to being easy to teach and recall, the 30:2 compression-ventilation rate is designed to reduce the likelihood of hyperventilation, which impairs venous return as shown in a well-done animal study (103). Additionally, other data suggest that no ventilation is needed for the first few minutes of sudden cardiac arrest and need for initial ventilations has remained a matter of debate (9).

As previously noted, when the airway is secured, continuous chest compressions should be delivered continuously in an asynchronous mode with the ventilation. Unfortunately, the by-product of increasing the speed of chest compression is fatigue, detected by both animal (104) and human studies (105).

Acceptable Variations of Closed Chest Compressions

1. Interposed abdominal compression. The interposed abdominal compression (IAC) CPR technique uses a dedicated second rescuer to provide manual compression of the abdomen (midway between the xiphoid and the umbilicus) during the relaxation phase of chest compression. The purpose is to enhance venous return during CPR (106-108). When IAC CPR performed by trained providers was compared with standard CPR for in-hospital cardiac arrest, IAC CPR improved ROSC and short-term survival in two randomized trials and improved survival to hospital discharge in one study (108,109). The implementation of this technique is limited by the extensive complex training needed and the need for at least three rescuers to provide coordinated compressions and ventilation.

2. Active Compression-decompression. Active compression-decompression CPR (ACD CPR) is performed with a handheld device equipped with a suction cup to actively lift the anterior chest during decompression. It is thought that decreasing intrathoracic pressure during the decompression phase enhances venous return to the heart. Although substantial animal data demonstrated that the device improves cardiac output during CPR, clinical trials reported mixed results, with some showing improved survival (110-112) and others finding no effect when compared to standard CPR (113-116). The reason for the disparate results is not clear, but some data suggest that extensive training is required to use this device effectively, and there is a higher rate of rescuer fatigue from the increased effort required during both compression and relaxation. At the time of this writing, no ACD CPR device is approved by the Food and Drug Administration (FDA) for sale in the United States.

3. Phased thoracic-abdominal compression-decompression. This technique is essentially a combination of the IAC CPR and ACD CPR techniques described above. A handheld device is used to alternate chest compression and abdominal decompression with chest decompression and abdominal compression. The utility of this technique is also limited by the need for a dedicated rescuer and the complexity of training (117).

Mechanical Adjuncts for Circulation

1. Mechanical piston device. This device is composed of a plunger mounted on a backboard, which is driven by compressed gas to depress the sternum. The rate and depth of compressions is adjustable with a 50% compression cycle. Use of this device resulted in improved end-tidal CO2 in both in-hospital and out-of-hospital settings (118-120). The expense and bulkiness of the device has limited its widespread distribution and use.

2. Vest device (load distributing band). The load-distributing band (I-LDB) is a circumferential chest compression device composed of a pneumatically or electrically actuated constricting band and backboard. The band is wrapped around the patient and compresses the entire chest, producing
thoracic pump CPR. Use of this device improved survival to emergency department arrival in one study (121); an FDA-approved device (AutoPulse) is available. A cohort study conducted before and after introduction of the load-distributing band found an improved rate of ROSC and survival to hospital discharge (122), whereas a randomized multicenter trial reported at the same time found no survival benefit 4 hours after the arrest and a lower rate of survival to hospital discharge (123).

**Advanced Life Support**

Advanced life support (ALS) entails advanced airway management including use of ancillary equipment to support ventilation and oxygenation, prompt recognition, and, when appropriate, treatment of life-threatening arrhythmias using electrical therapy including defibrillation, cardioversion, pacemaker insertion, and pharmacologic therapy. ALS includes the use of pharmacologic therapy and advanced procedures extending into the postarrest setting such as the use of therapeutic hypothermia.

**Advanced Airway Management**

**Tracheal Intubation.** Endotracheal intubation is indicated when the rescuer is unable to adequately ventilate or oxygenate the arrested or unconscious patient with bag-mask ventilation, or if prolonged ventilation is required and airway protective reflexes are absent in the patient with a perfusing rhythm. A properly placed endotracheal tube (ET) is the gold standard method for securing the airway. It maintains a patent airway to deliver effective positive pressure ventilation and use of positive end-expiratory pressure when indicated. It possibly reduces the risk from aspiration, especially if a cuffless tube is used, and it provides a means to suction airway secretions and provides an alternate route for resuscitation drugs delivery (124). No randomized adult studies have compared the outcome from endotracheal intubation to bag-mask ventilation, but one prospective randomized controlled trial in children showed no survival benefit of endotracheal intubation over bag-mask ventilation by trained EMS personnel (125). Attempted endotracheal intubation by less skilled rescuers results in complications. Retrospective studies reported a 6% to 14% incidence of misplaced or displaced ETs (126–128).

To minimize the risk of esophageal intubation, it is important to use an end-tidal CO₂ (ETCO₂) detector or esophageal detector device in addition to careful auscultation and observing symmetric chest rise. In patients requiring movement, exhaled CO₂ monitoring (capnography) is helpful to detect tube displacement. Proper tube position should be confirmed by chest radiography when feasible. The intensivist is expected to master the art of intubation. However, expert personnel may not be immediately available in the ICU. Due to the technical difficulty and skill level needed for endotracheal intubation, there is increased emphasis on learning bag-mask ventilation, which could be used primarily for ventilation and can serve as rescue backup if attempted endotracheal intubation fails.

**Confirmation of Correct Endotracheal Tube Placement.** Clinical signs used to confirm correct ET placement are (a) visualization of bilateral chest rise, (b) bilateral breath sounds over the lateral lung fields, (c) absent breath sounds over the epigas-

**ETCO₂ detector device.** A disposable colorimetric device that detects ETCO₂ has been investigated as a guide to correct endotracheal tube placement (129–131). The device fits on the end of the ET, and is normally purple in color; exhaled CO₂ turns the color to bright yellow indicating that the ET is in the trachea. This device has high sensitivity for esophageal placement of ETs in both cardiac arrest and patients with a perfusing rhythm (133–135), but poor specificity for tracheal placement. The device may suggest esophageal placement when the tube is in the trachea in patients who are morbidly obese, in late pregnancy, and with pulmonary edema or severe airway obstructive disease (e.g., asthma). These false-positive events occur because either the trachea collapses (obesity and late pregnancy) or air is not easily recruitable from the lung (pulmonary edema and severe airway obstruction). Sensitivity and specificity of EDDs are poor in children younger than 1 year old, and they are not recommended in children weighing less than 20 kg (136).

**Advanced Airway Adjuncts.** Multiple airway adjuncts are available in cases of failed endotracheal intubation and difficult bag-mask ventilation. Two of these devices, the laryngeal mask airway and esophageal tracheal Combitube, are widely used and discussed in depth in Chapter 38 (Airway Management). These adjuncts are "rescue airways," a bridge rather than alternate to endotracheal intubation that is readily mastered with appropriate training. Although they facilitate effective ventilation, they do not allow tracheal suctioning or provide reliable airway protection.

**Electrical Therapy**

Electrical therapy is one of the mainstays of ALS, especially in adults. Electrical energy is used to treat life-threatening cardiac...
Defibrillation. Defibrillation is defined as delivery of electrical energy resulting in termination of VF for at least 5 seconds following the shock (145,146). The goal is to quickly depolarize the whole myocardium, terminating the rhythm and hoping that a sinus rhythm will start. Defibrillation should not be confused with ROSC or survival. In most adults, the initial postshock rhythm is asystole or an organized slow rhythm without a pulse (i.e., PEA). This observation is the basis for the current guideline recommendation to immediately begin chest compressions after shock delivery.

Defibrillator device. There are two types of defibrillator devices available: (a) manual defibrillator devices require the rescuer to analyze the rhythm and then manually set and deliver the shock. The device is selected and delivered to the victim, and then the rescuer is responsible for ensuring good contact and delivering the correct energy dose with the first shock. (b) Automatic defibrillator devices analyze the rhythm, determine if a shock is required, and deliver the shock if needed automatically. There are two types of automatic defibrillators: internal implantable cardioverter defibrillator (ICD) and automated external defibrillator (AED). Defibrillators are also characterized by the mode and waveform of electrical current delivered into monophasic and biphasic defibrillators. Animal and human data show that biphasic defibrillators have a higher first shock success in terminating VF compared with monophasic devices; the latter are no longer produced. Manufacturers use various methods of delivering the biphasic energy, and more recently, triphasic and quadruphasic defibrillators are being developed and appear to be potentially more effective than biphasic defibrillators (147,148).

Monophasic defibrillators deliver current sequentially in one direction or polarity during one shock. In the first phase, the current moves from one paddle to the other as with monophasic defibrillators, and during the second phase, the current flow reverses direction. Biphasic defibrillators also can be further classified based on the waveform changes into (a) biphasic truncated exponential waveform and (b) rectangular biphasic waveform. Several randomized trials (149-151) showed that defibrillation with biphasic waveforms of relatively low energy (200 J) is safe and has equivalent or higher efficacy for termination of VF than monophasic waveform shocks of equivalent or higher energy, which decreases the risk of myocardial injury and increases shock efficacy.

Defibrillation dose. The optimal energy dose for the first shock of subsequent shocks required for effective defibrillation remains unknown despite multiple studies (152,153). The ideal shock dose for a biphasic device is one that falls within the range documented as effective using that specific device. It is reasonable to use selected energies of 150 J to 200 J with a biphasic truncated exponential waveform or 120 J with a rectangular biphasic waveform for the initial shock. For second and subsequent biphasic shocks, the same or higher energy can be given. The rescuer should have knowledge of the type of defibrillator being used and select the energy dose accordingly. Most manual defibrillators are set to an initial default of 200 J of energy. This is not necessarily the optimal dose in all cases, but was selected because it falls within the reported range of doses effective for first and subsequent biphasic shocks. If only a monophasic defibrillator is available, then an energy dose of 360 J is recommended for all shocks.

During shock, the current delivered is the key determinant of successful defibrillation (153–155). Current delivery is determined by the energy level selected and by the patient’s trans-thoracic impedance. The current delivered is the energy divided by the impedance, or current = energy / impedance.

The optimal dose for effective defibrillation in infants and children is not known. The upper limit for safe defibrillation is also not known, but doses more than 4 J/kg (as high as 9 J/kg) have effectively defibrillated children (159,160) and pediatric animal models (161) with no significant reported adverse effects. Recommended manual defibrillation (monophasic or biphasic) doses for children are 2 J/kg for the first attempt and 4 J/kg for subsequent attempts.

Electrode position. Either handheld paddles or self-adhesive pads are used for shocks. Electrodes are applied to the bare chest in the conventional sternal-apical (anterolateral) position. The right (sternal) chest pad is placed on the victim’s right superior-anterior (infraclavicular) chest and the apical (left) pad is placed on the victim’s inferior-lateral left chest, lateral to the left breast. Other acceptable pad positions are placement on the lateral chest wall on the right and left sides (baxillary) or the left pad in the standard apical position and the other pad on the right or left upper back (avoiding the scapular bone). Similar positions are recommended for children, particularly the anterior-posterior position since many defibrillators have only a single size electrode pad and there should be at least 1 inch between the pads. The rescuer should make sure that electrodes are not overlapping and are not on top of implanted devices or transdermal medicine patches if present. If the patient is wet, the chest should be wiped dry before placement. Another caution is to avoid placing the oxygen source near the electrodes, particularly in children where the distance from the face to the chest is short.
Electrode size. In general, the largest pad or paddle that can be placed on the chest while avoiding contact between the pads or paddles should be used. There should be at least 1 inch between the pads. Paddles that are too small increase the risk of skin burn injury.

Automatic rhythm analysis and automatic external defibrillators. AEDs are computerized compact devices that use voice and visual prompts to guide lay rescuers and health care providers to safely defibrillate VF or pulseless VT (139,162–164). In a large prospective randomized trial (165) funded by the AHA, the National Heart, Lung, and Blood Institute (NHLBI), and several AED manufacturers, lay rescuer CPR-AED programs in targeted public settings doubled the number of survivors from out-of-hospital VF SCA when compared with programs that provided early EMS call and early CPR. Despite limited evidence, AEDs should be considered within the hospital as a way to facilitate early defibrillation (a goal of <3 minutes from collapse), especially in areas where staff have limited to no rhythm recognition skills or defibrillators are used infrequently.

For children 1 to 8 years of age, the rescuer should use a pediatric dose-attenuator system with the AED if one is available (160). If the rescuer provides CPR to a child in cardiac arrest and does not have an AED with a pediatric attenuator system, the rescuer should use a standard AED.

Electrical Cardioversion. Electrical cardioversion is used for some life-threatening arrhythmias causing rapid cardiovascular deterioration. These include VF and supraventricular tachycardia (SVTs), such as paroxysmal atrial tachycardia (PAT), atrial flutter, or atrial fibrillation with a rapid ventricular response. The electrode size and positioning is the same as that used in defibrillation; the difference between cardioversion and defibrillation is described below.

Technique. Unlike defibrillation, cardioversion must be synchronized with the patient's electrocardiogram (ECG). The ideal discharge point is during the upstroke of the R wave of the QRS complex. Delivery of the energy during the T wave of the QRS may result in ventricular fibrillation. Most commercially available defibrillators automatically coordinate the discharge to the patient's ECG if the machine is placed in the synchronized mode and the QRS complex is of adequate size for consistent detection. If the defibrillator does not "sense" the QRS complex, increasing the ECG gain improves sensing. Cardioversion should never be attempted with quick-look paddles, because ECG artifact may make synchronization impossible. Unsyncronized cardioversion should only be used when the available equipment does not allow synchronization.

Energy level. The amount of energy recommended for emergency cardioversion varies with the rhythm (166,167); 100 J is recommended for atrial fibrillation and 50 J for atrial flutter (168). Monomorphic ventricular tachycardia responds well to cardioversion, and 100 J should be attempted initially. Pulseless ventricular tachycardia behaves like ventricular fibrillation, and 200 J should be used initially. For cardioversion in conscious patients, sedation with intravenous diazepam, midazolam, or methohexitol is indicated, and the cardioversion accomplished with the lowest energy possible (50–200 J). In children, the recommended initial cardioversion dose is 0.5 to 1 J/kg.

External Cardiac Pacing. While, in general, external (transcutaneous) pacing is not recommended for patients in asystolic cardiac arrest, it is our opinion that it should be always considered in the ICU or other critical care areas of the hospital where the device and adequate skill are promptly available. However, three randomized controlled trials (169,170) of fair quality indicated no improvement in the rate of admission to hospital or survival to hospital discharge when paramedics or physicians attempted to provide pacing in asystolic patients in the prehospital or hospital (emergency department) setting. Pacing can be considered in patients with symptomatic bradycardia when a pulse is present.

Pharmacologic Therapy

Pharmacologic therapy in CA is used to increase the rate of ROSC and terminate or limit the risk of recurrent arrhythmias. It is recognized that there are no placebo-controlled studies showing that any vasopressor during human CA increases survival to discharge; indeed, some studies questioned the benefit of their use (171). For obvious reasons, it is unlikely that a large prospective randomized study on this issue will ever be available. However, drug administration is now a secondary priority during CPR with primary emphasis placed on high-quality chest compressions with minimal hands-off time. Since there is little evidence for their use, the number of pharmacologic agents recommended during CPR decreased.

Route of Administration for Resuscitation Medications. It is important to remember that a central venous line may not be available at the time of the arrest, nor is its immediate placement necessary to ensure survival. Peripheral intravenous access can be used effectively with the advantage of not interrupting CPR. When only a peripheral vascular access is available, it is crucial to rapidly follow the medication bolus with a 10- to 20-mL fluid bolus to ensure central delivery. If obtaining any venous access is difficult despite numerous attempts, intravenous (IO) cannulation is an alternative for drug delivery. Studies in both children (172,173) and adults (174) suggest the effectiveness of this route in all age groups. Commercially available kits are available for easy use.

If IV or IO access cannot be achieved, then some resuscitation medications can be administered via instillation through an ET, if available. Lipid-soluble medications that can be delivered via ET are lidocaine, epinephrine, atropine, naloxone, and vasopressin. This route, however, results in much lower blood concentrations compared with IV administration. Optimal doses of the medications delivered via the ET route are not known, but it is recommended to administer at least 2 to 3 times the IV recommended doses. In fact, animal data suggest that using standard intravenous epinephrine doses via the ET route may not achieve high enough plasma concentrations to be effective (175,176).

Once vascular access is achieved, resuscitation drugs should be immediately readministered if cardiac arrest is still present.

Epinephrine. Epinephrine is the most commonly used medication during CPR. Epinephrine’s primary action in CA is to increase the coronary perfusion pressure through systemic vasoconstriction mediated by its α-adrenergic effects. The
β-adrenergic effects are relatively unimportant. Indeed, even when complete β-adrenergic blockade is used in an animal cardiac arrest model, epinephrine is effective, whereas α-adrenergic blockade completely eliminates epinephrine’s effects (177).

Epinephrine is used primarily during CA due to asystole and PEA. It is a second-line agent used for shock-refractory VF or pulseless VT. Although there are no clinical trials demonstrating that epinephrine is effective in CA, there is substantial animal data and anecdotal clinical experience that epinephrine elevates the coronary perfusion pressure and thus myocardial blood flow. There are little pharmacologic data supporting the currently recommended dose of 1 mg of epinephrine in adult cardiac arrest and 0.01 mg/kg in children. Epinephrine should be given intravascularly whenever possible since intratracheal doses are erratically absorbed as noted above. If only the endotracheal route is available, the dose should be increased to 2 to 2.5 mg in adults and 0.1 mg/kg in children.

Since epinephrine’s beneficial effect is through systemic vasoconstriction, it seems reasonable to speculate that higher doses would produce a greater increase in coronary and cerebral perfusion pressure. This hypothesis was supported by initial animal data (178), but subsequent animal studies (179,180) and clinical trials in adults (181,182) and children (183) failed to show a beneficial effect. Indeed, clinical experience suggests that high-dose epinephrine is harmful rather than beneficial and is only indicated in clinical conditions characterized by poor adrenergic responsiveness, such as severe septic shock and β-blocker, neuromuscular blockade, or systemic bupivacaine overdose.

Vasopressin. Vasopressin is an endogenous antidiuretic hormone that, when given at high doses, causes vasoconstriction by directly stimulating vascular smooth muscle V1 receptors (184). Vasopressin improves coronary perfusion pressure, but unlike epinephrine, offers theoretical advantages of cerebral vasoconstriction, possibly improving cerebral perfusion. Its lack of β1-adrenergic activity potentially avoids unnecessary increases of myocardial oxygen consumption, resulting in postresuscitation arrhythmias. Vasopressin has a longer half-life of 10 to 20 minutes compared to the 3 to 5 minutes observed with epinephrine.

Several human studies suggested that vasopressin achieved a comparable ROSC in CA, although no additional benefit was seen compared with epinephrine (185,186). A recent meta-analysis showed a trend favoring vasopressin (187) compared to epinephrine. Recent guidelines continue to recommend epinephrine as the agent of choice, but one dose of vasopressin 40 units IV/IO is now indicated as an alternative to replace the first or second dose of epinephrine in all CA including asystole and PEA.

Vasopressin is not currently recommended for pediatric use since in an animal model of pediatric asphyxia CA, ROSC was higher in the epinephrine group (188). Vasopressin is contraindicated in conscious patients with ischemic heart disease as it may provoke angina. Other adverse effects include increased mesenteric and renal vascular resistance, bronchial constriction, and uterine contractions in women.

Sodium bicarbonate. Metabolic and respiratory acidosis develops during CA resulting from anaerobic metabolism, leading to lactic acid generation and inadequate ventilation along with reduced blood flow during CPR, which leads to inadequate pulmonary delivery of carbon dioxide for elimination. Untreated acidosis suppresses spontaneous cardiac activity, decreases the electrical threshold required for the onset of ventricular fibrillation, decreases ventricular contractile force, and decreases cardiac responsiveness to catecholamines such as epinephrine. An elevated PCO2 tension probably is more detrimental to myocardial function and catecholamine responsiveness than metabolic acidosis. CO2 readily diffuses across myocardial cell membranes, causing intracellular acidosis and resulting in life-threatening derangements of myocardial function. Likewise, cerebrospinal fluid acidosis may occur secondary to the diffusion of CO2 across the blood-brain barrier, producing postasphyxial cerebral acidosis. Since sodium bicarbonate buffering of excess protons transiently increases the PCO2 tension, sodium bicarbonate administration without sufficient ventilation and circulation to remove the CO2 that it produces seems to be more detrimental than helpful (189,190). Sodium bicarbonate therapy may be useful after interventions such as defibrillation, cardiac compression, intubation, ventilation, and more than one trial of epinephrine have been used. Some evidence suggests that any benefit from bicarbonate therapy may be related more to volume expansion from its high sodium content rather than its buffering effects (191).

If arterial blood gas and pH measurements are not available, the recommended initial dose of sodium bicarbonate is 1 mEq/kg intravenously; half of this dose may be repeated at 10-minute intervals. For pediatric patients, the 1 mEq/kg dose should be diluted 1:1 with sterile water to reduce the osmolality.

Administration of excessive amounts of sodium bicarbonate can result in metabolic alkalosis, leftward shift of the oxyhemoglobin dissociation curve, interference with tissue oxygenation, hypernatremia, hyperchloremia, and worsening of respiratory and myocardial acidosis if adequate ventilation and perfusion cannot be achieved and maintained.

Atropine. Atropine is used during CPR for its vagolytic actions. A reduced vagal influence on the heart improves both the rate of sinoatrial node discharge and impulse conduction through the atrioventricular (AV) conduction system, with a resulting increase in heart rate. Atropine is used in sinus bradycardia when accompanied by hypotension or frequent premature ventricular contractions (PVCs) secondary to unsuppressed ectopic electrical activity arising in the area of injured tissue during the prolonged period after resuscitation. Sinus bradycardia after myocardial infarction may predispose the heart to the onset of ventricular fibrillation (192). When profound bradycardia is present, acceleration of the heart rate above 60 beats per minute (bpm) may improve cardiac output and reduce the incidence of ventricular fibrillation. Atropine also may be useful for treating high-degree AV block with a slow ventricular rate and asystole occurring after increased parasympathetic tone that results in suppression of the electrical activity to the heart (193).

The dosage of atropine for severe symptomatic bradycardia is 0.5 to 1.0 mg intravenously repeated every 3 to 5 minutes until the desired pulse rate is obtained or a maximum of 0.04 mg/kg has been given. A larger dose has little therapeutic value, and a smaller dose may actually slow the heart rate. Endotracheal dose is 2 to 2.5 mg. Although not routinely recommended in the treatment of asystole, when used, incremental doses of
Lidocaine. Lidocaine decreases ectopic electrical myocardial activity by raising the electrical stimulation threshold of the ventricle during diastole. In ischemic myocardial tissue after infarction, it may suppress re-entrant arrhythmias such as ventricular tachycardia or fibrillation. There is now good evidence, however, that other agents are superior to lidocaine in terminating VT (194,195); hence, it is not considered a first-line agent. The 2005 guidelines recommend lidocaine only when amiodarone is not available.

Lidocaine may be used in stable monomorphic VT and polymorphic VT with normal or prolonged QT interval if ventricular function is not decreased.

The loading dose of lidocaine is approximately 1 to 1.5 mg/kg given as an intravenous bolus. If needed, repeat 0.5 to 0.75 mg/kg every 5 to 10 minutes, up to a total of 3 mg/kg, followed by a continuous infusion of 30 to 50 μg/kg/minute (1–4 mg/minute in a 70-kg patient) (192).

Excessive doses may induce heart block or depression of sinus node discharge, especially in patients with pre-existing conduction disturbances. Toxicity may occur in oliguric or anuric patients because renally excreted lidocaine degradation products also have pharmacologic effects and toxic potential. Early signs of lidocaine toxicity are due to central nervous system (CNS) effects and include anxiety, loquacity, tremors, metallic taste, and tinnitus. These may be followed by somnolence, respiratory depression, apnea, and, in severe cases, cardiovascular collapse. If CNS irritability occurs, lidocaine therapy should be withdrawn and a barbiturate or a benzodiazepine may be administered if deemed necessary and if the patient's circulatory status is sufficiently stabilized.

Procainamide. Procainamide suppresses both atrial and ventricular arrhythmias with similar mechanisms of action to those of lidocaine. It may suppress an ectopic irritative focus and blocks re-entrant arrhythmias by slowing electrical conduction. Procainamide may be superior to lidocaine in terminating VT (196).

Procainamide is used in the management of PVCs, ventricular tachycardia, and persistent ventricular fibrillation, but amiodarone is usually preferred.

Incremental bolus injections of procainamide are slowly infused at 20 mg/minute until (a) the arrhythmia is controlled, (b) hypotension occurs, (c) the QRS complex is widened 50% from baseline, or (d) a total dose of 17 mg/kg (1.2 g in a 70-kg adult) is given followed by a continuous infusion of 1 to 4 mg/minute to prevent recurrent arrhythmias. Other effective administration schedules have been tested and approved; all are designed to maintain a therapeutic plasma level of 4 to 8 μg/mL.

Procainamide can have profound myocardial depressant effects and may block after myocardial infarction; therefore, continuous ECG and arterial blood pressure monitoring are mandatory. End points of therapy include hypotension and a greater than 50% widening of the QRS complex.

Amiodarone. IV amiodarone is a complex drug with effects on sodium, potassium, and calcium channels on myocardial cells as well as α- and β-adrenergic blocking properties. The 2005 guidelines denote that it is the preferred agent for both atrial and ventricular arrhythmias, especially in the presence of impaired cardiac function.

Amiodarone is recommended for narrow-complex tachycardias that originate from a re-entry mechanism (re-entry SVT); ectopic atrial focus; control of hemodynamically stable VT; polymorphic VT with a normal QT interval, or wide-complex tachycardia of uncertain origin (195,197); and control of rapid ventricular rate due to accessory pathway conduction in pre-excited atrial arrhythmias with AV nodal blockade in patients with preserved or impaired ventricular function (198).

In the treatment of arrhythmias with a pulse, 150 mg amiodarone is given IV over 10 minutes, followed by a 1 mg/minute infusion for 6 hours and then a 0.5 mg/minute maintenance infusion over 18 hours. Supplementary infusions of 150 mg can be repeated every 10 minutes as necessary for recurrent or resistant arrhythmias to a maximum manufacturer-recommended total daily IV dose of 2.2 g. One study found amiodarone to be effective in patients with atrial fibrillation when administered at relatively high doses of 125 mg/hour for 24 hours (total dose 3 g) (199).

When used in the treatment of VF or pulseless VT, a bolus dose of 300 mg is recommended diluted in 20 to 30 mL of D5W. A single second dose may be given (150 mg) in 3 to 5 minutes for shock-refractory VF or VT. In children, 5 mg/kg is given as a rapid bolus and may be repeated up to 15 mg/kg.

The major adverse effects of amiodarone are hypotension and bradycardia, which can be prevented by slowing the rate of drug infusion. In addition, amiodarone can increase the QT interval, and therefore its use should be carefully considered when other drugs that can prolong the QT interval are administered.

Calcium. Calcium ion plays a critical role in myocardial contractility and action potential generation, but studies have shown no benefit of calcium in CA (200,201), and therefore calcium probably should not be considered during CA only when acute hyperkalemia or hypocalcemia is suspected or calcium channel blockers were administered. The use of calcium during CA is controversial because of the fear that it may produce a tetanic contraction of an irritable myocardium or depression of the sinus node, resulting in asystole. Since maintenance of the 10,000-fold higher extracellular calcium concentration compared with the intracellular concentration is energy dependent, exposing hypoxic-ischemic cells to high plasma calcium concentrations may result in calcium overload, which activates a number of toxic mechanisms. This effect is particularly worrisome in the brain because it is so sensitive to hypoxia.

When indicated, the recommended dose is 5 to 10 mL of a 10% solution of calcium chloride (8–16 mg/kg). A bolus may be repeated at 10-minute intervals, if necessary. Calcium salts cannot be mixed directly with sodium bicarbonate because it precipitates as calcium carbonate. Several calcium preparations are available for intravenous use: Calcium chloride and calcium gluconate are the most popular. Calcium gluconate is given in a dose of 6 to 8 mL if peripheral IV access is available. Undiluted calcium chloride given through a peripheral vein may cause sclerosis and tissue injury; therefore, if a central site is not available and the patient is not in cardiac arrest, it should
either be diluted or calcium administered in a less irritating form (e.g., calcium gluconate). In children, a dose of 10 to 20 mg/kg of calcium chloride is recommended.

Rapid administration of a large bolus of calcium chloride, especially through a central venous catheter, may produce severe sinus bradycardia or sinus arrest. Calcium must also be used cautiously in patients receiving digitals, because it can produce or accentuate digitalis toxicity.

Magnesium. Magnesium is recommended for the treatment of torsades de pointes VT with or without cardiac arrest. It has not been shown to be helpful during non-torsades de pointes VT (202,203). Although magnesium is a calcium channel blocker and theoretically may protect ischemic cells from overload, there are no data supporting its routine use in cardiac arrest. If needed, magnesium is given at a dose of 1 to 2 g diluted in D5W over 10 to 60 minutes. In children, a dose of 25 to 50 mg/kg is used.

Rapid administration may result in hypotension and bradycardia. Magnesium also should be used cautiously in patients with renal failure.

Algorithms for Advanced Life Support
The treatment algorithms of pulseless cardiac arrest and tachycardia with pulse are summarized in Figures 46.2 and 46.3, respectively.

Monitoring during Cardiopulmonary Resuscitation

Clinical
There are no reliable clinical indicators of the effectiveness of CPR provided. Although feeling for a pulse during chest compressions is used clinically to assess the effectiveness of chest compressions, no study has validated this technique. Moreover, data show that a palpated femoral pulse may be from retrograde venous pulsation rather than antegrade arterial flow (204).

Hemodynamics
When invasive arterial and central venous pressure monitoring are present, coronary perfusion pressure (CoPP) monitoring is possible. It is calculated as CoPP = aortic diastolic pressure minus the right atrial pressure (CoPP = DRP – RAP). A CoPP > 15 mm Hg is predictive of ROSC (193,205) and increased CoPP correlates with improved 24-hour survival in animal studies (206).

Arterial Blood Gas
The acidosis gradient between arterial and central venous blood (largely determined by PCO2) reflects the effectiveness of blood flow during low-flow states such as CPR (207). Similarly, animal studies and case reports report similar arteriovenous differences in PCO2 between arterial and venous blood at the organ level (e.g., heart) and the entire organism (208–210). These studies suggest that measurement and comparison of a venous and arterial blood gas may be predictive during cardiac arrest. Furthermore, it suggests that the arterial blood gas does not accurately reflect the severity of tissue acidosis, hypoxia, or hypercarbia during both in-hospital and out-of-hospital cardiac arrest. Instead, the arterial blood gas often reflects the effectiveness of ventilation during cardiac arrest. Similarly, both animal and human data show a significant worsening of the arterial blood gas values for acidosis and oxygenation with ROSC, representing the washout of built-up tissue acids.

End-tidal Carbon Dioxide
Aerobic and anaerobic cellular metabolism generates CO2, which rapidly diffuses out of the cell and into tissue capillaries, is transported to the lungs, is exhaled, and can be measured as end-tidal CO2 (211,212). Under normal conditions, end-tidal PCO2 is 2 to 5 mm Hg less than the PaCO2 (211,212). Several studies of end-tidal carbon dioxide during CPR typically reflect changes in the effective pulmonary blood flow (211,212). Under conditions of constant minute ventilation, end-tidal CO2 is linearly related to cardiac output, even during extremely low blood flow rates (213), and is therefore useful clinically as a monitor of perfusion during shock and CPR. Indeed, a number of studies showed that end-tidal CO2 varies directly with cardiac output during cardiac arrest (214–216) and provides a useful indicator of the efficacy of resuscitation efforts. Not surprisingly, ETCO2 also predicts outcome in adults (217–220) and children (221). Additionally, capnography is used in resuscitation research as an indication of pulmonary blood flow, which serves as a proxy for the direct measurement of cardiac output (210,222,223).

Following the administration of epinephrine, end-tidal CO2 typically falls, suggesting that the relationship of end-tidal CO2 to cardiac output is altered, but in fact it is maintained and reflects a global fall in cardiac output due to changes in pulmonary and peripheral vascular resistance and preferential redirection of blood flow (219). When used in cardiac arrest, epinephrine decreases pulmonary blood flow (i.e., overall cardiac output) and end-tidal CO2 while increasing coronary perfusion pressure and myocardial blood flow because of increased peripheral vascular resistance (224).

Coronary artery perfusion pressure also correlates closely with end-tidal CO2 (218,223). Several studies of end-tidal carbon dioxide during low blood flow states found that levels changed significantly with changes in minute ventilation (226). When minute ventilation doubled, end-tidal carbon dioxide decreased by 50%, and when minute ventilation decreased by 50%, end-tidal carbon dioxide doubled. Thus, end-tidal carbon dioxide varies inversely with minute ventilation and can be used to monitor ventilation during low-flow conditions. If both perfusion and ventilation are changed, end-tidal carbon dioxide levels can be difficult to interpret.

Postresuscitation Care
After ROSC, the risk of mortality remains high for cardiac arrest patients as most deaths happen during the first 24 hours following successful resuscitation (227,228). Postresuscitation care is one of the most important elements of CPR that can potentially result in improved outcome (229). The goals of postresuscitation care are to (a) optimize cardiovascular and neurologic support, (b) identify the possible cause of arrest...
Cardiac arrest

Asystole/
PEA

 VF/VT

Continue CPR for 5 cycles
Obtain IV/IO
Give 5 cycles of CPR
Consider 6 Hs 5 Ts (Table 46.1)
Give ONE shock
Manual biphasic 120–200 J
AED device specific
Monophasic 360 J
Resume CPR immediately after shock for 5 cycles
Repeat every 3–5 minutes
May give vasopressin 40 U to replace first or second dose of epinephrine
Consider atropine 1 mg for slow PEA rate
Repeat every 3–5 minutes (up to 3 doses)
Give 5 cycles of CPR
Consider 6 Hs 5 Ts (Table 46.1)
Give ONE shock, as above
Resume CPR immediately after shock for 5 cycles
Obtain IV/IO
Epinephrine 1 mg when CPR restarted
Repeat every 3–5 minutes
May give vasopressin 40 U to replace first or second dose of epinephrine

Obtain IV/IO
Epinephrine 1 mg when CPR restarted
Repeat every 3–5 minutes
May give vasopressin 40 U to replace first or second dose of epinephrine

Consider when CPR started
Amiodarone 300 mg IV/IO and then additional 150 mg
 Lidocaine 1–1.5 mg/kg first dose then additional 0.5–1 mg/kg (max 3 mg/kg)
 Magnesium 1–2 g for torsades

Consider 6 Hs 5 Ts (Table 46.1)

FIGURE 46.2. Suggested treatment algorithm for pulseless cardiac arrest. It is important that resuscitation drugs should be given during the cardiopulmonary resuscitation (CPR) cycles to minimize “no CPR” time. BLS, basic life support; PEA, pulseless electrical activity; VF, ventricular tachycardia; VT, ventricular fibrillation; AED, automated external defibrillator; IO, intraosseous. (Adapted and modified from 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2005;112[24]:IV1-IV-211.)

and prevent recurrence, and (c) use measures that may improve long-term neurologic outcome.

After ROSC, vital signs, clinical and laboratory signs of tissue perfusion, the occurrence of cardiac arrhythmias, and neurologic status should be frequently monitored. Obtain appropriate intravenous and intra-arterial vascular access as needed to monitor the patient and obtain frequent laboratory studies. Obtain an arterial or central venous blood gas (preferably both) to assess for the presence of acid-base abnormalities, and obtain electrolytes including magnesium, phosphorus, ionized
Section IV: Essential Physiologic Concerns

Tachycardia with pulses

Start BLS, oxygen; attach monitor
Check rhythm, blood pressure, oximetry
Consider reversible causes (Table 46.1) at each step

Immediate synchronized cardioversion
- Obtain IV access
- Consider sedation if patient is conscious
- Consider expert consultation
- If becomes pulseless, go to Figure 46.3

Check instability
- Altered mental status
- Low blood pressure or other signs of shock
- Chest pain
- Poor capillary refill

Obtain IV access
Obtain 12-lead ECG (when available) or rhythm strip
QRS duration?

VT or uncertain rhythm
- Amiodarone 150 mg IV over 10 minutes; repeat as needed to maximum dose of 2.2 g/24 hours
- Consider elective synchronized cardioversion

SVT with aberrancy
- Follow regular narrow QRS algorithm (see §)

Atrial fibrillation with aberrancy
- Follow irregular narrow-QRS algorithm (see §)
- Pre-excited atrial fibrillation (WPW)
  - Expert consultation advised
  - Avoid AV nodal blockers (e.g., adenosine, digoxin, diltiazem, verapamil)
  - Consider antiarrhythmics (e.g., amiodarone)
- Recurrent polymorphic VT
  - Torsades de pointes
  - Magnesium 1-2 g over 5-60 minutes, then infusion

Conversion to sinus rhythm?
- Consider expert consultation

Re-entry supraventricular tachycardia
- Continue monitoring
- Treat recurrence
  - Adenosine
  - Longer-acting AV nodal blocking agents
    - Diltiazem
    - β blockers

Likely atrial flutter, ectopic atrial tachycardia, or junctional tachycardia
- Ventricular rate control
- β blockers
- Treat underlying cause
- Consider expert consultation

Calcium, and blood urea nitrogen (BUN) to ensure that there are no electrolyte abnormalities and to help assess renal function. The gradient between arterial and venous oxygen saturation can help evaluate the adequacy of tissue perfusion. Trained personnel with resuscitation equipment must accompany the patient during transport to an appropriate monitoring unit.

It is increasingly recognized that postresuscitation care has an important effect on ultimate survival following cardiac arrest. Studies are currently evaluating optimal treatments for
postresuscitation care (230). The following are generally accepted as part of postresuscitation care.

**Glucose Control**

Many studies showed a strong association between hyperglycemia after cardiac arrest and poor neurologic outcome (28,231–233). Hyperglycemia developing after cardiac arrest likely reflects the effects of epinephrine and endogenous stress hormones as well as reduced glucose metabolism by the injured brain. What is not clear, however, is whether control of the glucose concentration following cardiac arrest results in an improved outcome. Data from studies in adult surgical and medical critical care patients showed reduced mortality with tight glucose control (234,235). Based on these data in critically ill patients, it seems reasonable to normalize the glucose concentration in patients after cardiac arrest, but the evidence for this approach is lacking. Furthermore, it is not clear if glucose should be restricted or if insulin should be used to normalize the glucose concentration, but the general approach is to frequently monitor the glucose concentration and use an insulin infusion to normalize the glucose concentration if it is elevated.

The target range for glucose concentration in this situation is not known, but a range of 80 to 110 mg/dL is commonly used. The risk of this approach is that hypoglycemia may occur, resulting in further injury to at-risk brain cells.

**Respiratory System**

Some respiratory dysfunction can be present after ROSC, and patients require ongoing mechanical ventilation and oxygen administration. ET position should be assessed upon arrival to the critical care unit. Ventilator support should be adjusted to help normalize the patient’s acid–base status and work of breathing, while recognizing that high respiratory rates and airway pressure may impair venous return and thus cardiac output. If an end-tidal CO\(_2\) monitor is available, ventilation rate should be adjusted to maintain end-tidal CO\(_2\) within a range of about 35 mm Hg to 45 mm Hg. In patients with obstructive pulmonary disease and increased resistance to exhalation, a lower ventilation rate should be used (e.g., six to eight breaths per minute) to prevent air trapping and to allow enough time for complete exhalation (32). If more severe injury is present, it is usually due to pulmonary edema, gastric content aspiration, or acute respiratory distress syndrome (ARDS). While generally in these cases oxygenation may be improved by the application of positive end-expiratory pressure, more detailed treatment guidelines are reviewed in other sections of this textbook.

**Cardiovascular System**

Cardiac function and rhythm can be altered after cardiac arrest.

This results from both global ischemia and reperfusion injury post ROSC or by the effect of defibrillation itself, which can cause myocardial stunning (236,237). Hemodynamic instability is common after cardiac arrest and may be multifactorial including a combination of cardiac dysfunction, altered capillary permeability, variations in venous and arterial vascular tone, and the elaboration of cytokines that may mimic sepsis (238). Invasive hemodynamic monitoring and judicious use of inotropes with vasopressor or vasodilators properties may be needed (227).

Although post–cardiac arrest arrhythmias may be detrimental, prophylactic treatment may also produce adverse effects, so prophylactic antiarrhythmics are not recommended. If an antiarrhythmic was used to achieve ROSC, then it should be continued, although the selection of antiarrhythmic agents for longer-term use is best made in consultation with a cardiologist.

**Central Nervous System**

After ROSC, the brain often experiences an initial brief increase in blood flow (“hyperemia”) phase, which is followed by a significantly reduced (“no-reflow phenomenon”) phase due to microvascular dysfunction and thrombosis (239,240). In addition, excitatory neurotransmitters are released, increasing metabolic demand in vulnerable areas of the brain such as the hippocampus. In general, efforts are focused on optimizing cerebral perfusion pressure by treating hypotension and maintaining, when feasible, a 20% to 30% higher than normal mean arterial pressure, especially in cases at risk for increased intracranial pressure. Fever and seizures can be seriously detrimental to the brain and should be aggressively treated. There are insufficient data to recommend for or against the use of prophylactic anticonvulsants in postcardiac arrest.

**Temperature Control**

Avoiding Hyperthermia. Temperature dysregulation may be present after ROSC, which may result from brain injury as shown in animal models of cardiac arrest (241–244). Fever is associated with worse neurologic outcome in humans after cardiac arrest or ischemic brain injury (245,246). Antipyretics and surface cooling techniques should be used to achieve normothermia. Some experts recommend routine administration of antipyretics, and the 2005 guidelines recommend frequent or continuous temperature monitoring and aggressive treatment of fever in the postarrest setting.

**Hypothermia after Cardiac Arrest.** In 2002, two prospective randomized controlled clinical trials of therapeutic hypothermia were conducted in comatose survivors after cardiac arrest in adults. In the Australian study (247), survivors of out-of-hospital VF cardiac arrest with coma were randomized in the field by EMS providers to hypothermia or normothermia, with cooling beginning in the prehospital setting. Cooling was maintained for 12 hours followed by passive rewarming. Good outcome (survival and good functional recovery) was observed in 21 of 43 (49%) hypothermia patients versus 9 of 34 (26%) normothermic patients (p = 0.046; number needed to treat [NNT] = 5.95; 95% confidence interval [CI], 2.1–84). A larger European trial in a similar patient population (248) showed favorable neurologic outcome at 6 months with the use of 24 hours of therapeutic hypothermia begun after hospital arrival following out-of-hospital ventricular fibrillation cardiac arrest (75 of 136 [55%]) compared with the normothermic group (54 of 137 [39%]). The risk ratio for this study was 1.40 (95% CI, 1.08–1.81) and the NNT was 7 (95% CI, 3.6–25). Subsequently, the ILCOR recommended the use of therapeutic hypothermia for adult patients who were comatose following ventricular fibrillation cardiac arrest and suggested that hypothermia should be considered in comatose patients with other rhythms after out-of-hospital cardiac arrest (249).

Several neonatal trials also showed a beneficial effect of 72 hours of therapeutic hypothermia following perinatal asphyxia (250,251), but there are no clinical data in children to
extracorpooreal Life Support and Cardiopulmonary Resuscitation

Rapid use of extracorporeal membrane oxygenation (ECMO) in the context of CPR is termed extracorporeal cardiopulmonary resuscitation (ECPR). This technique is invasive and requires emergent cannulation of a large central vein and artery. Venous blood is passed through a membrane oxygenator for oxygenation and removal of CO₂, and returned in an artery to augment blood circulation. The setup and personnel required for this procedure are available in few large tertiary care centers with an in-house multidisciplinary team available to rapidly prepare the ECMO circuit. ECPR has been used in both pediatric and adult patients with primarily cardiac disease with a reported survival of 64% in neonates, 50% in children, and 51% in adults (256,257). In a large single-center report (258), 64 children who failed initial advanced life support were placed on ECMO during active CPR with the median duration of chest compressions of 50 minutes (range 5–105 minutes); remarkably, 33 (50%) survived for 24 hours and 21 (33%) survived to hospital discharge. Five of the survivors were noted to have significant neurologic impairment at hospital discharge. Use of extraordinary measures like ECPR and ECMO seems reasonable if the patient’s condition is reversible and ECMO can be established before irreversible brain damage happens during the cardiac arrest. One advantage of extracorporeal circulatory support is that it is relatively easy to rapidly cool the patient and maintain the target temperature for brain protection.

ETHICAL CONSIDERATIONS

Termination of Cardiopulmonary Resuscitation Efforts

CPR is inappropriate when survival is not expected. Even in the ICU, CPR can be administered by rescuers who may not know the patient or the advanced directive status of the patient. There are few criteria that can reliably predict the futility of CPR. Therefore, it is recommended that all patients with cardiopulmonary arrest receive CPR with the following exceptions:

1. There is a valid Do Not Attempt Resuscitation (DNAR) order, which can be verified.
2. Signs of irreversible death are already present (e.g., rigor mortis, decapitation, decomposition, dependent lividity).
3. No physiologic benefit can be expected with further CPR.
4. In newborns, gestational age or congenital abnormality is related to almost certain early death.

In the ICU, CPR is started without a physician’s order, which is based on implied consent for emergency treatment. As a minimal standard, it is expected that all nursing staff in the unit are able to immediately start BLS and be knowledgeable on the use of an automatic external defibrillator. However, the decision to stop CPR should be made by a physician treating the patient, ideally familiar with the patient’s pre-existing conditions. Although many factors have been studied as a guide to determine the futility of CPR, such as the presence of comorbidities, time to CPR, time to defibrillate, and initial rhythm, none of these either alone or in combination reliably predicts chances of neurologically intact survival (259,260). On rare occasions, good outcome with prolonged CPR has been reported (261–263). It is acceptable to prolong resuscitative efforts in children with recurrent VF or VT, drug toxicity, or a primary hypothermic insult.

Family Presence during Cardiopulmonary Resuscitation

It has been a common practice to exclude family of the arrest victim during CPR efforts. Helping the family of a dying patient is an important part of CPR and should be done in a compassionate manner considering the family’s cultural and religious beliefs and practices. Although there is concern that allowing family presence may become disruptive, interfere with resuscitation, cause them to experience syncope, and increase the risk of legal liability (264–266), these concerns were not substantiated by good evidence, and in the authors’ experience these factors are not a major issue. Family surveys following CPR suggested that the majority of family members want to be present during CPR (267,268). It is also suggested that being present at the time of death was comforting and helped with adjustment later (269). In pediatric arrest, most parents surveyed wanted an option to be present during CPR (270). The resuscitation team should be sensitive to the family’s presence and a team member should be assigned to comfort the family, answer questions, and clarify the procedures.

Future Considerations

Current Performance during Cardiopulmonary Resuscitation

Although U.S. and international guidelines continue to recommend high-quality CPR to improve survival from cardiac arrest (271), disturbing evidence seems to indicate that this is not happening in most cases. Studies suggest that bystander CPR
in witnessed cardiac arrest is performed in only about one third of the cases (272,273). There are several suggested reasons for this finding: CPR algorithms are complicated, training is inadequate, retention of skills is difficult, and both lay rescuer and professionals are afraid of transmission of diseases (274–276). An out-of-hospital cardiac arrest observational study in 176 adults showed that chest compressions were given only half of the available time during the resuscitation event. When CPR was adequate, compression time increased but still averaged 80% of the total resuscitation time (94). In the same setting (277), it was reported that police and firefighters performed CPR correctly only 45% of the time while waiting for ambulance personnel. Prehospital data indicate that most of the time “wasted” without CPR could be explained by programmed interruptions from automated defibrillators or pulse check. The quality of in-hospital CPR is also discouraging. Even highly trained hospital personnel such as ICU physicians often fall short of CPR guidelines during resuscitation efforts (95).

One other issue is the inability to completely relax the chest during the decompression phase (85). Recent laboratory data suggest that rescuer fatigue is an important cause of poor BLS CPR. Reduced performance due to fatigue was noted after just 1 minute and the magnitude was proportional to the compression speed. In a manikin model, an increased compression-to-ventilation ratio of 15:2 provided only 14.5% more compressions than a C:V ratio of 5:1, but nearly 81% of the rescuers perceived that a C:V ratio of 15:2 was more tiring than a 5:1 ratio (104). The quality of compressions seems to decrease significantly after 1 minute and then again between 4 and 5 minutes of CPR, suggesting the need for frequent rescuer rotations, between 1 and 5 minutes, to optimize the effectiveness of chest compression. Interestingly, subjective awareness of fatigue is usually present only after 5 minutes, despite the substantial decrease of chest compression depth at 1 minute (278). Decay of chest compression quality always includes rate and depth.

Studies have shown that the decay of skill mastery is almost immediate after a CPR class with a trend toward too shallow compressions by lay rescuers (279) and too deep compressions by health professionals (280). In general, repeated manikin training is more successful in correcting too shallow compressions than too deep (280). Another study revealed that most trained personnel provided too much ventilation, ~10 breaths/minute, during out-of-hospital cardiac arrest. Hyperventilation increases mean airway pressure, resulting in decreased venous return and cardiac output, which worsens survival (40).

These devices improve ETCO2 and quality of CPR in both in-hospital and out-of-hospital settings (98,281–283).

Medical Emergency Teams

Recently, there is increased emphasis on the use of a dedicated in-hospital rapid response system (RKS) for early intervention in at-risk patients. There are two components of this system: (a) case recognition and team activation, and (b) the emergent medical team. Medical emergency teams (METs) are the response component of an RKS that has the capability of prescribing therapy, providing advanced airway management, securing central venous access, and beginning ICU-level care at the bedside (284). These teams are often composed of experienced nurses and doctors with skills in critical care who are available 24 hours a day to seek out, assess, and manage patients who clinically pose a threat to develop cardiac arrest. Several small studies documented reduction in cardiac arrest rates with the use of METs (285,286), but a recent large clustered randomized study was not able to show any difference in cardiac arrest, unexpected death, and unplanned ICU admissions with the use of METs (287). Further studies are needed to evaluate the effectiveness and feasibility of METs.

Research Considerations

Research in patients with CA is extremely challenging. Most of the interventions need to be applied very quickly to be beneficial, which makes it hard for researchers to obtain institutional review board approval.

Ethical Considerations and Informed Consent

Obtaining consent from family is not always possible or practical at the time of cardiac arrest. Some researchers argue that presuming consent in these patients serves a benefit to the living and others argue that since the patient is clinically dead, consent is unnecessary since a dead patient is without autonomy. This argument does not account for the potential emotional harm to the family, who may oppose use of a deceased loved one for research, and also does not account for their cultural and religious beliefs.

Recently the importance of CPR research was recognized and the FDA adopted regulations that allow exception for consent in certain circumstances. Obtaining an exception requires the researcher to consult with experts in the field and representative lay persons, along with open and full public disclosure of the study, including the methodologic details of need, for, and benefit of the research.

## Training Needs for Quality Improvement

### Involvement of Lay Rescuers

Few advanced life support interventions improve survival of SCA patients. It is recognized that early and effective CPR and defibrillation are effective. Therefore, there is a need to increase access to CPR education and improve CPR training quality and retention of skills.

### Feedback Devices

Use of devices that can help rescuers by prompting or giving voice feedback or tone may increase the effectiveness of CPR. References

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93. Dickinson ET, Vindal VP, Schneider BM, et al. Effectiveness of mechanical


